



NOTES

COMMA-SHAPED RODS

MICROBE OVERVIEW

- Gram-negative, facultative anaerobes, motile, non-spore forming

COMMA-SHAPED RODS SUMMARY

SPECIES	HOST	MODE OF TRANSMISSION	PATHOLOGY
CAMPYLOBACTER JEJUNI	Wild, domestic birds, mammals	Fecal-oral Uncooked meat (esp. poultry) Unpasteurized milk	Africa, Middle East
HELICOBACTER PYLORI	Humans	Unknown	Gastric, peptic ulcers; chronic gastritis
VIBRIO CHOLERAE	Humans Fish	Fecal-oral Contaminated food/water	Cholera

CAMPYLOBACTER JEJUNI

osms.it/campylobacter-jejuni

PATHOLOGY & CAUSES

Characteristics

- Zoonotic disease
 - Reservoir in wild, domestic mammals, birds (esp. poultry)
- **Oxidase +**; invasive; microaerophilic; sensitive to heat, desiccation, acidity, irradiation, disinfectants

Virulence factors

- Fimbriae-like filaments
 - Promote attachment to intestinal

epithelial cells

- Possesses single, unsheathed flagellum in one end (monotrichous)/two flagella each at both ends (amphitrichous)
 - Provide motility, chemotaxis (mucin is chemoattractant → tropism for ileum, colon, rectum)
- Surface proteins (eg, PEB1, CadF) promote colonization, invasion of intestinal epithelial cells
- Lipopolysaccharide (LPS)
 - Plays role in adherence, evasion of host immune response (undergoes antigenic variation)

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Culture

- **Isolation specimen:** stool, food
- **Media:** blood/charcoal agar with microaerophilic atmosphere (5–10% O₂, 3–5% CO₂), thermophilic environment (optimal 42°C/107.6°F)
- *C. jejuni*: one of most common bacterial causes of gastroenteritis with acute diarrhea

Transmission

- **Fecal-oral**, contaminated water

Pathogenesis

- Incubation period 1–7 days; tropism for distal ileum, colon, rectum
- Inoculation of bacteria → passage through upper GI tract → colonization, adherence to surface epithelium of distal ileum, colon → non-inflammatory secretory diarrhea (exact mechanism unknown)
- Invasion of intestinal epithelium, proliferation → release of cytolethal distending toxin → cell damage, inflammatory response → **dysentery** with fecal leukocytes
- **Rare:** translocates into lamina propria, spreads to mesenteric lymph nodes (mesenteric adenitis) → extraintestinal infections (e.g. meningitis, cholecystitis, UTI)
 - Occurs mostly in immunocompromised

RISK FACTORS

- **Consumption of undercooked meat/ unpasteurized milk**
- Underlying conditions/medications that reduce/buffer gastric acidity (e.g. proton pump inhibitors)
- Individuals with HIV/AIDS

COMPLICATIONS

- Toxic megacolon, massive bleeding, colonic perforation
- **Reactive arthritis**
- Associated with **Guillain-Barré syndrome**
 - Sialic acid contained bacterial core oligosaccharide can resemble gangliosides → cross-activation of autoreactive T/B cells (molecular mimicry)

SIGNS & SYMPTOMS

- Vary in severity depending upon inoculum concentrations; range from asymptomatic carriage to systemic illness
 - Most episodes mild, self-limiting (up to one week); rarely persists up to several weeks
- Fever, myalgia, malaise, headache (early symptoms, 1–2 days); severe periumbilical abdominal pain, cramping, secretory, inflammatory **diarrhea**, vomiting
 - Abdominal pain may mimic acute appendicitis
 - **Secretory diarrhea:** more common in children
 - **Inflammatory diarrhea:** tenesmus, bloody stools, fecal leukocytes

DIAGNOSIS**LAB RESULTS**

- Stool culture
- Rapid diagnosis with carbolfuchsin stain/ phase-contrast/dark-field microscopic examination of fresh stool specimen in case of acute manifestation
- PCR-based methods, enzyme immunoassays (EIAs) directly from stool

OTHER DIAGNOSTICS

- Clinical manifestations
- Histology
 - Acute mucosal inflammation with edema, cellular infiltration of lamina propria, crypt abscess formation

TREATMENT**MEDICATIONS**

- Antibiotics for severe cases, immunocompromised individuals
 - Erythromycin/azithromycin

OTHER INTERVENTIONS

- Replacement of fluids, electrolytes

HELICOBACTER PYLORI

osms.it/helicobacter-pylori

PATHOLOGY & CAUSES

Characteristics

- Urease +, catalase +, oxidase +; non-invasive
- Microaerophilic
 - Requires oxygen, lower concentrations than present in atmosphere

Virulence factors

- Possesses 2–7 unipolar sheathed flagella (H-antigen)
 - Provide motility, chemotaxis (sense pH, move bacteria towards beneficial environment)
- Lipopolysaccharide (LPS)
 - Promotes adherence, causes inflammation
- Coccoid form
 - More resistant form; occurs as adaptation to hostile environment outside human body
- Urease
 - Important for survival, colonization
- Mucolytic enzymes
 - Allow passage through mucus layer to gastric epithelium
- Adhesive proteins (Hop proteins)
- Vacuolating cytotoxin A (VacA)
 - Damages epithelial cells; disrupts tight junctions, causes apoptosis
- Cytotoxin associated gene CagA (CagA)
 - Triggers inflammation
- Type IV secretion system
 - Pili-like structure for injection of effectors (e.g. CagA)
- Proteases, lipases
- Biofilm formation

Culture

- Isolation specimen: vomitus, diarrheal stools
- Media: blood agar/selective Skirrow's media

incubated at 37°C/98.6°F in 5% oxygen; small, uniformly sized, translucent bacterial colonies

- *H. pylori*: causative agent of most common chronic infection in humans; common cause of duodenal, gastric ulcers, chronic gastritis

Transmission

- Unknown; fecal/oral, oral/oral transmission suggested
- Reservoir
 - Human (majority of cases); found in primates in captivity, domestic cats, sheep
- Also found in municipal water in endemic areas of infection with polymerase chain reaction (PCR) techniques

Pathogenesis

- Bacterial urease hydrolyzes gastric luminal urea to form ammonia → ↑ gastric pH → formation of protective layer around bacteria → survival in hostile gastric environment
- ↑ pH → mucin liquefies → *H. pylori* passes through mucous layer to surface epithelium via bacterial flagella, mucolytic enzymes → attaches to specific gastric epithelial cell receptors via surface adhesins (Hop proteins) → release of proteases (VacA, CagA) + host immune response → inflammation, tissue injury
- Disruption of mucous layer → susceptibility to acid peptic damage
- Chronic inflammation, tissue injury together with acid peptic damage → chronic gastritis, peptic ulcers (10–20% risk)

RISK FACTORS

- Low socioeconomic status
- Increased housing density
- Lack of running water
- Genetic susceptibility
- Swimming in pools, rivers, streams

COMPLICATIONS

- **Gastric carcinoma** (1–2% risk): chronic gastritis → atrophic gastritis, intestinal metaplasia, carcinoma
 - Chronic inflammation, ↑ TNF, ↑ IL-6, ↑ bacterial proteases → excessive tissue damage, cell mutation → intestinal metaplasia → carcinoma
- Gastric **mucosa-associated lymphomas** due to persistent immune stimulation of gastric lymphoid tissue

SIGNS & SYMPTOMS

- Majority of cases asymptomatic
- Acute infection
 - Upper abdominal pain, nausea, loss of appetite
- Chronic infection
 - **Chronic gastritis**: upper abdominal pain, nausea, bloating, vomiting/melena (black stool)
 - **Peptic ulcers**: stomach pain/ache; occurs with empty stomach, between meals, early morning

DIAGNOSIS

LAB RESULTS

- Blood antibody test
- Stool antigen test
- Carbon **urea breath test**
 - Individual ingests ^{14}C - or ^{13}C -labelled urea, which bacterium metabolizes, yielding labelled carbon dioxide detectable in breath
- Urine enzyme-linked immunosorbent assay (ELISA) test

TREATMENT

MEDICATIONS

- One-week “**triple therapy**”: antacid/acid-reducing drugs (H₂-receptor antagonists/ **proton pump inhibitors**) + two antibiotics
 - Bismuth salicylate + metronidazole + tetracycline
 - Ranitidine bismuth citrate + tetracycline + **clarithromycin/metronidazole**

- Omeprazole/pantoprazole + clarithromycin, **amoxicillin**: in case of penicillin sensitivity, replace amoxicillin with **metronidazole**

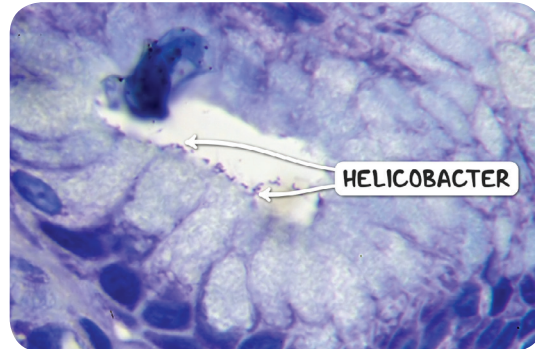


Figure 65.1 *Helicobacter* organisms in a gastric pit.

VIBRIO CHOLERAЕ (CHOLERA)

osms.it/vibrio-cholerae

PATHOLOGY & CAUSES

Characteristics

- **Oxidase +**; non-invasive; halophilic; genome consists of two circular chromosomes; sensitive to acid, drying
- **Reservoir**: aquatic environments (saltwater, brackish)
- **Fermentation**: glucose, sucrose

Virulence factors

- **Cholera toxin**
 - Only toxigenic strains; responsible for pathogenesis of massive, watery diarrhea; coded by filamentous bacteriophage (CTXΦ)
- Other toxins that increase mucosal permeability
 - Zona occludens toxin (ZOT), accessory cholera enterotoxin (ACE), WO7 toxin 17
- Lipopolysaccharide (LPS)
 - > 200 serogroups; O1, O139 associated with cholera epidemics
- Motility
 - Single polar flagellum (H-antigen)
- Toxin-coregulated pilus (TCP)
 - Present only in toxigenic strains; promotes adherence, aggregation of bacteria; coded by genes in Vibrio pathogenicity island (VPI)
- Mucinase
 - Digests mucous layer of gastrointestinal (GI) tract

Culture

- **Isolation specimen**: stool, rectal swab
- **Media**: thiosulfate citrate bile salts sucrose (TCBS) agar/taurocholate tellurite gelatin agar (TTGA); large, yellow colonies (2–4mm diameter) with opaque centers, translucent edges

- *V. cholerae*: diverse species; pathogenic, toxin-producing (toxigenic) variants cause cholera
- Cholera characterized by **profound secretory diarrhea** → rapid, life-threatening dehydration
- Transmitted by fecal-oral route/contaminated food or water

Pathogenesis

- Inoculation → passage through upper GI tract → rapid movement of bacteria through mucous via flagellum → colonization of small intestine via TCP → releases cholera toxin
- Cholera toxin (AB protein toxin) → B subunit binds to GM1 ganglioside on intestinal epithelial cell, allows entry of A subunit → **activates G-protein regulated adenylyl cyclase** → **↑ intracellular cyclic adenosine monophosphate (AMP)** → **secretion of chloride, sodium**; inhibition of sodium chloride absorption → **massive fluid secretion**; loss of sodium, chloride, bicarbonate, potassium

TYPES

Pathogenic (toxin-producing), non-pathogenic

Serological classification: O antigen differences

- Serogroup O1 is subdivided into two serotypes (Inaba, Ogawa), two biotypes
 - *El Tor*: cause of current global pandemic of cholera
 - *Classical*: cause of previous *V. cholerae* pandemics; now thought extinct
- Serogroup O139
- Non-O1/O139

RISK FACTORS

- Travel to endemic/epidemic areas
- **Inadequate access to clean water**

- Shellfish consumption in areas with sporadic cholera
- People with blood group O at higher risk for severe cholera (mechanism unknown)

COMPLICATIONS

- If untreated
 - Dehydration, hypovolemic shock in 4–12 hours, death in 18 hours to several days
- Renal failure secondary to hypovolemia
- Electrolyte imbalances
 - Hypokalemia, metabolic acidosis
- Pneumonia (esp. in children) due to aspiration of vomit

SIGNS & SYMPTOMS

- Incubation period is 28–48 hours
- Asymptomatic to severe depending upon strain, inoculum concentration ($\geq 10^8 \rightarrow$ severe form)
- Abrupt onset of profound watery diarrhea (grey, cloudy, flecked with mucus; “rice-water stool”); painless, without tenesmus; loss of 1 liter of fluid per hour in severe cases
- Moderate to severe vomiting, borborygmus, abdominal discomfort
- Dehydration
 - Thirst, dry mucous membranes, decreased skin turgor, sunken eyes, hypotension, weak/absent radial pulse, tachycardia, tachypnea, hoarse voice, oliguria
- Altered mental status
 - Somnolence, restlessness, lethargy

DIAGNOSIS

LAB RESULTS

- Hypoglycemia/hyperglycemia
- Hypercalcemia, hypermagnesemia, hyperphosphatemia
- $\uparrow$ hematocrit due to volume depletion

OTHER DIAGNOSTICS

- Clinical presentation
- Microbiologic diagnosis

- Culture on TCBS agar
- Dark field microscopy/dipstick test of stool specimen for rapid confirmation in non-endemic areas (detectable in stool for 1–2 weeks without antimicrobial therapy)

TREATMENT

MEDICATIONS

- Oral antibiotic treatment reduces duration, severity of disease
 - Doxycycline for adults
 - Azithromycin for children, pregnant individuals

OTHER INTERVENTIONS

- Prophylaxis
 - **WC-rBS (Dukoral) vaccine:** monovalent inactivated oral cholera vaccine containing killed whole cells of *V. cholerae* O1, additional recombinant cholera toxin B subunit
 - **BivWC (Shanchol) vaccine:** bivalent inactivated oral vaccine containing killed whole cells of *V. cholerae* O1, O139
 - **CVD 103-HgR/Vaxchora vaccine:** attenuated oral vaccine derived from serogroup O1 classical Inaba strain
- Rapid, aggressive **volume replacement** (oral/intravenous fluids)
- Adequate nutrition to prevent malnutrition
- Correction of electrolyte imbalances
- Zinc supplementation reduces duration, severity of disease